REVIEW ARTICLE JPAR 2024

Journal of Pharmaceutical Advanced Research

(An International Multidisciplinary Peer Review Open Access monthly Journal)

Available online at: www.jparonline.com

A review on Allergic rhinitis with little insights of Diagnostic procedure and management

Vigneswaran R*, Natarajan P, Thiruppathi M, Ganesh H, Kodishwaran R

Department of Pharmacology, Sankaralingam Bhuvaneswari College of Pharmacy, Anaikuttam, Sivakasi – 626130, India.

Received: 15.03.2023 Revised: 22.03.2024 Accepted: 26.03.2024 Published 31.03.2024

ABSTRACT: Among atopic diseases, allergic rhinitis (AR) is the most prevalent and widespread type. It is a relatively prevalent illness that peaks in adolescence and affects people of all ages. Numerous causal factors, such as dust mites, molds, pollens, and animal dander, have been associated with allergic reactions (AR). When inhaled allergens interact with IgE antibodies on airway cells, allergic rhinitis occurs. Itchy palate; itchy, red, and watery eyes; sneezing; watery rhinorrhea; and nasal congestion are all signs of allergic rhinitis and conjunctivitis. Edema and venous congestion of the nasal mucosa produce a feeling of pressure in the sinuses, a cough, and blockage of the eustachian tubes. Even though allergic rhinitis is not a life-threatening condition, it is clinically relevant since it is a major risk factor for inadequate asthma control, impacts quality of life and productivity at work or school, and lies at the basis of numerous challenges. The history and physical examination are usually sufficient to make the diagnosis. When a patient has symptoms of refractory rhinitis, computed tomography most often reveals sinusitis, and spirometry is helpful in identifying subclinical asthma. It is essential to manage allergic rhinitis according to guidelines. An essential part of treatment is educating the patient.

Corresponding author:
Dr. Vigneswaran R
Assistant professor
Sankaralingam Bhuvaneswari College of Pharmacy,
Anaikuttam, Sivakasi – 626130, India.
Tel: +91-7708316032
E. Mail ID: vignesvicky985@gmail.com

Keywords: Allergic rhinitis, allergy, pollen, dust, IgE, sneezing, Hypersensitivity, Anti-allergic.

INTRODUCTION:

Sneezing, itching, rhinorrhea, and/or nasal congestion are some of the nasal symptoms that can indicate a diverse illness called rhinitis. Postnasal discharge and other symptoms affecting the eyes, ears, and throat are often present with rhinorrhea. Both adults and children can get rhinitis for a variety of reasons. Allergies cause around half of all occurrences of rhinitis. When allergens trigger rhinitis, the immune system's gamma globulin E (IgE)-mediated reaction to particular allergens like dust mites, molds, pollens, and animal dander causes inflammation that result in symptoms. The immune response includes cell invasion and activation to the nasal mucosa, as well as the release of inflammatory

mediators [1]. Social interactions, academic achievement, and productivity at work are all negatively impacted by allergic rhinitis, especially in more severe cases [2]. The effects of rhinitis on academic achievement are negative [3]. As a primary factor in both diagnosis and treatment, quality of life is highlighted in the ARIA (Allergic Rhinitis and its Impact on Asthma) guideline. It offers a comprehensive, practical, evidence-based, systematic strategy to treating allergic rhinitis. The GRADE (grading of recommendations assessment, development, and evaluation) technique has been used in recent years to update and review the treatment plan [4]. The anti-inflammatory effects of intranasal corticosteroids on various cell types make them the most effective class of drugs; certain compounds, even in children, do not exhibit systemic bioavailability when used for extended periods. Currently, sublingual and subcutaneous immunotherapies are available, mostly for people whose allergic rhinitis cannot be controlled with medication or by avoiding certain allergens. Additionally, immunotherapy is the only medication that is currently available that likely modifies the course of the disease, slowing the progression of both rhinitis and sensitization to asthma [5].

EPIDEMIOLOGY:

There are 400 million cases of allergic rhinitis worldwide, and the ailment is more common in industrialized nations, especially those with English-speaking populations. Bostock described seasonal allergic rhinitis in the UK for the first time in 1819 and provided more details four years later [6]. According to epidemiologic studies conducted across different nations, the prevalence of rhinitis varies between 3 and 19 %. The majority of the studies that have provided the most information indicate that 10 to 20 % of people have perennial rhinitis and 10 % of people have SAR (hay fever) [7]. In the US, 20 to 40 million people suffer from allergic rhinitis [8]. In the general population, AR appears to be occurring more frequently. According to studies conducted by the Swedish army, the prevalence of hay fever improved from 4 to 8 % during the ten-year period between 1971 and 1981. In Tucson, Arizona, atopic skin test reactivity also increased from 39 to 50 % during the course of eight years of testing [9]. There also seems to be an increase in the prevalence of AR among children. In one study, 42 % of children aged 6 had AR that was identified by a doctor. According to a recent Finnish study, the incidence almost doubled between 1979 and 1991. As of the present, allergic reactions are the most prevalent allergy disease and one of the most common chronic disorders in children [10].

ETIOLOGY:

The atypical disposition to produce particular IgE in reaction to common and harmless environmental allergens is known as atopy. Atopic dermatitis, asthma, allergic rhinoconjunctivitis, and food allergies are examples of atopic disorders that typically run in families. Numerous genetic loci on chromosomes 2, 5, 6, 7, 11, 13, 16, and 20 have been associated with atopy [11]. High socioeconomic status, environmental pollution, being born during a pollen season, not having older siblings, entering nursery or preschool later than the age of four, heavy maternal smoking during the first year of life, exposure to indoor allergens like dust mites and animal dander, elevated serum IgE concentrations (>100 IU/mL before the age of six), positive skin prick tests for allergens, and early introduction of foods or formula are further risk factors for allergic rhinitis [12]. Heavy alcohol use in adulthood may also be a risk factor. The findings of multiple investigations have demonstrated that early exposure to a variety of infectious agents, including lipopolysaccharides, endotoxins, hepatitis A, Mycobacterium species, and *Toxoplasma gondii*, as well as their products, protects against the development of atopy. This finding supports the hygiene hypothesis's guiding principles [13].

CLASSIFICATION:

Initially, allergic rhinitis has been classified as either seasonal or perennial based on the person's sensitivity to allergens throughout the year allergens such as dust mites, cockroaches, animal dander, molds, or seasonal pollens. This plan fails globally because many affected people have both seasonal and perennial allergen sensitization, yet seasons do not exist in many parts of the globe.

Seasonal allergic rhinitis:

Seasonal allergies include outdoor mold spores and pollens from grass, trees, and weeds. Usually, the symptoms develop during a specific season when the outside air is high in aeroallergens. The place of residence determines how long a person is exposed to certain allergies during the season. Therefore, the disorder is simpler to diagnose if one is familiar with the primary trees, grasses, and weeds in the area and their pollination season [14]. There is seasonal variation

observed in some outdoor mold spores, with summer and fall seeing the largest concentrations [15].

When exposed to pollen, common symptoms include frequent allergy symptoms of the eyes, itching, and sneezing, as well as a sudden onset of abundant watery rhinorrhea. Although it does happen, congestion is typically not the most bothersome symptom. Generally, seasonal pollen counts correspond with the start and resolution of symptoms. Even so, when the pollen season ends, hyper responsiveness to irritating triggers, which arises from the inflammatory response of the late phase and stimulation responses often persists. These triggers include exertion, changes in temperature, undesirable flavours, and tobacco smoke.

Perennial allergic rhinitis:

Eosinophils, mast cells, TH2 lymphocytes, and macrophages infiltrate the tissue and cause persistent tissue edema when exposed to dust mites, cockroaches, indoor molds, and pet dander throughout the year. In locations where pollen is consistently present, [16] PAR can also be caused by pollen.

There isn't a single, widely recognized definition for chronic rhinitis. The most common definition of it is a disease that causes two or more of the following symptoms and lasts longer than nine months per year: sneezing paroxysms, nasal blockage due to enlarged nasal mucosa and serous or seromucus hypersecretion. The majority of patients mostly experience symptoms of nasal congestion and mucus production (postnasal drip); sneezing, itching, and watery rhinorrhea may be infrequent [17].

Perennial allergic rhinitis with seasonal exacerbations:

Depending on the range of allergen sensitivity, AR symptoms may also be chronic, with seasonal exacerbations [18].

DIAGNOSIS:

When diagnosing AR in children, a thorough history and physical examination are the most useful diagnostic tools [19]. A greater understanding of the ailment and any possible comorbidities is essential for an accurate and prompt diagnosis in pediatric patients. Children's AR is frequently misdiagnosed or left untreated, mistaken for other conditions, including persistent colds. It's possible to misdiagnose AR as "cough-variant" asthma when coughing is the main symptom, particularly at night. At every well-child visit, the clinician should ask specific questions about the presence and cause of rhinitis symptoms, be aware of the symptoms and signs of rhinitis, and be familiar with the differential diagnosis of allergic reactions in children in order to make an efficient and precise diagnosis [20]. Evidently, rhinorrhea, congestion, sneezing, and itching are common signs of allergic reactions (AR). Bilateral, unilateral, or continuous side-to-side congestion is all possible. Generally speaking, it is more conspicuous at night. Snoring can be a night time symptom of nasal blockage, and mouth breathing is likely to occur in the patient. Therefore, irregular sleep patterns could be a sign of an allergy condition [21]. The medical history of a patient can provide information about other allergic disorders, allergenic triggers, and a family history of allergies [22]. Specific IgE reactivity to airborne allergens (related to the patient's history) must be confirmed, either by skin-prick testing or by recording specific IgE in serum, in order to validate an allergic rhinitis diagnosis. Additionally, this testing gives information that can be used to guide allergy-specific immunotherapy or environmental management strategies [23]. In most regions of the world, inhaling allergens of which house dust mites and grass and tree pollens are the most common causes of allergic rhinitis [24]. Numerous nasal pathology abnormalities are associated with non-allergic rhinitis, and further focused research may be required. The term occupational rhinitis refers to a variety of pathological abnormalities (caused by both allergic and non-allergic processes) that are all related to the development of symptoms after exposure to work [25].

MANAGEMENT OF ALLERGIC RHINITIS:

The following aspects of an effective therapeutic strategy for allergic rhinitis include immunotherapy consideration, medication, and patient education regarding allergen or irritant avoidance [26].

Patients' Education:

A lot of individuals do not realize how dangerous their persistent, irritating runny, itchy noses and sneezing could be. If someone thinks they might have a cold, they could skip going for medical attention and instead choose to self-treat with over-the-counter medications [27]. In addition to the unsatisfactory results of over-the-counter medications, these hazards include drowsiness, decreased motor control, and cognitive impairment. Using learning tools, the patient should be informed about allergic rhinitis, its consequences, and therapy once they have been officially diagnosed by their

primary care physician [28]. It is necessary that the prescribed regimen be followed, and giving written directions is crucial in this regard [29]. It is an important objective to include the patient's family in the team when caring for the patient with problematic allergic rhinitis [30].

Pharmacotherapy:

The two main groups of medications used to treat allergic rhinitis symptoms are intranasal corticosteroids and oral H1 antihistamines (Table 1). Depending on the patient's reaction to therapy and the prevalent symptoms, these medications may be administered in combination or as monotherapy. For certain patients, other medications like cromolyn sodium might be more suitable.

Table 1. Differential to use nasal and oral drugs on Allergic rhinitis.

Class of Drug	Medication
Nasal Treatment	
Corticosteroids	Sprays—fluticasone, mometasone, ciclesonide, triamcinolone, flunisolide, beclametasone
Antihistamines	Azelastine, olopatadine
Chromones	Sodium cromoglicate, nedocromil sodium
Anticholinergics	Ipratropium bromide
Decongestants	Ephedrine, pseudoephedrine, xylometazoline
Oral Treatment	
Antihistamines	Second-generation antihistamines-levocetirizine and cetirizine, desloratadine and loratadine, fexofenadine, acrivastine, rupatadine, carebastine and ebastine
Corticosteroids	Hydrocortisone, prednisolone
Antileukotrienes	Leukotriene receptor antagonists (montelukast and zafirlukast) and leukotriene synthesis inhibitors
Decongestants	Pseudoephedrine

Inadequate therapy for rhinitis is widespread, even with treatment guidelines available [31]. Compared to non-directed treatment, guidelines-directed management offers greater symptom control and an enhanced quality of life [32]. The most effective medication, which can be administered orally or topically, can be determined using the ARIA categorization [33]. Three meta-analyses [34] have demonstrated that intranasal corticosteroids are the most effective treatment agents for allergic rhinitis. They are either superior to or equal to the combination of an antihistamine and an antileukotriene [35]. For moderate-to-severe rhinitis, intranasal corticosteroids should be utilized, even in youngsters, for whom effective long-term safety evidence is available. About 79 % of adult patients with rhinitis in secondary care improved on intranasal corticosteroids [36]. Anti-inflammatory medication therapy of the nasal cavity decreases bronchial hyperreactivity linked to allergen exposure in seasonal rhinitis [37,38]. Anti-IgE monoclonal antibodies may help people with related allergic rhinitis and are approved for use in patients with severe asthma [39].

Individuals with allergic rhinitis are not recommended to use two pharmaceutical therapies. First, sedative antihistamines impair cognitive and occupational performance [40] and are linked to workplace and vehicular accidents. Second, systemic side effects, subcutaneous and muscle necrosis, and other potentially serious adverse events are linked to intramuscular corticosteroid injections [41]. Additional drug information is provided in Tables 1 and 2.

Immunotherapy:

Patients who have not reacted to medication, have severe symptoms, or whose symptoms are turned on by allergens for which strong extracts are available may benefit from immunotherapy [42]. Unlike medications, which suppress symptoms, immunotherapy tries to change the immune system and may be able to treat allergic rhinitis. When applied topically, subcutaneous immunotherapy reduces symptoms and medication requirements for patients with allergic rhinitis and appears to prevent asthma attacks and new stimulation [43]. Allergen extract injections are administered repeatedly as part of subcutaneous immunotherapy. It is only prescribed for patients with severe allergic rhinitis whose symptoms cannot be adequately managed with medication or whose drug side effects limit their options for treatment. Subcutaneous allergen immunotherapy works well, but less than 0.1 % of patients experience a slight but real risk of developing a systemic allergic reaction. Subcutaneous allergen immunotherapy should only be administered to patients in clinics run by medical professionals who are experienced in adjusting immunotherapy dosages. Patients must be monitored for 60 min following injection (or 30 min in the USA) due to the possibility of serious systemic side effects.

Table 2. Comparison of nasal and oral treatment for Allergic rhinitis.

Aspect	Nasal treatment	Oral treatment
Mechanism of Action	Targets local inflammation in nasal passages, reducing congestion, sneezing, and itching.	Acts systemically to inhibit histamine receptors, relieving symptoms such as sneezing and itching.
Route of Administration	Administered topically, directly to nasal mucosa via sprays or drops.	Taken orally, absorbed into the bloodstream for systemic distribution.
Onset of Action	Generally provides rapid relief, with effects often felt within hours.	Requires time for absorption and distribution, with onset typically within 30 min to 1 h.
Adverse Effect	Local irritation, dryness, or nosebleeds may occur. Prolonged use can lead to nasal mucosa atrophy.	Potential for systemic side effects, including drowsiness, dry mouth, and gastrointestinal upset.
Potential for Rebound Congestion	Minimal risk when used appropriately, but may occur with prolonged decongestant use.	Not applicable; rebound congestion is not a concern with oral treatments.
Suitability for Long Term Use	Generally safe for prolonged use under medical supervision, with careful monitoring for adverse effects.	May have limitations for extended use due to potential systemic side effects and corticosteroid toxicity.
Indicated Severity	Effective for mild to moderate symptoms, often used as first-line treatment.	Reserved for moderate to severe symptoms or cases unresponsive to nasal treatments.

Injections should only be administered in medical facilities with access to resuscitation equipment and specialists.

Both adults and children benefit from sublingual immunotherapy, which only requires physician monitoring for the first dose [44]. Because adverse effects are often limited to the upper respiratory tract and gastrointestinal tract and because there have only been a few reported cases of anaphylactic reactions without fatalities, it appears to be safer than subcutaneous immunotherapy [45]. Research indicates that the immunological and clinical advantages of sublingual immunotherapy remain for a period of three years of continuous treatment, much like the advantages of subcutaneous immunotherapy. Moreover, sublingual immunotherapy-specific local oral alterations are seen [46]. While more research is needed to determine lifespan and concordance, particularly in children [47], we are cautiously optimistic about sublingual immunotherapy as a successful asthma treatment and potential asthma preventer [48].

Surgery:

Not frequently is surgery required, unless it's necessary for topical nasal therapy in patients with severe septal deviations, turbinate hypertrophy, or nasal-valve malfunction that causes difficulty breathing via the nose. When medication treatment is ineffective for chronic rhinosinusitis, endoscopic sinus surgery may be necessary [49].

DISCUSSION:

A number of strategies for care are required for allergic rhinitis, which affects millions of people worldwide and is a significant clinical challenge. The identification of particular allergens that cause symptoms is a crucial component of the discussion, emphasizing the necessity of precise diagnostic instruments such as skin prick testing and particular IgE antibody assays [50]. The ongoing importance of pharmacological interventions in the treatment of allergic rhinitis is shown by recent reviews [51]. Confirm the effectiveness of antihistamines from the current generation, like desloratadine and cetirizine. Intranasal corticosteroids, such as fluticasone, are also essential for reducing inflammation and congestion in the nasal passages [52]. Moreover, the field of immunotherapy is constantly changing [53], which brings another level of individualized treatment by altering the immune response for long-lasting alleviation. A comprehensive therapy incorporates

patient education on environmental control measures, with a focus on allergy avoidance methods and the importance of nose irrigation, in addition to pharmacological interventions. The advancement of treatment methods and the improvement of patient outcomes depend on the continued investigation of novel medications and our ability to traverse the complexities of managing allergic rhinitis [54].

CONCLUSION:

Among atopic diseases, allergic rhinitis (AR) is the most common, affecting people of all ages and reaching its peak during adolescence. AR is caused by inhaled allergens that interact with IgE antibodies. It can cause a variety of symptoms, such as watery eyes, sneezing, nasal congestion, and palate itching. Although not fatal, its clinical significance is that it is a significant risk factor for insufficient asthma management, which affects people's general quality of life and productivity at work or school. In addition to a complete physical and medical history, refractory rhinitis patients may also benefit from the use of spirometry and computed tomography as part of the diagnosis procedure. Patient education plays a critical role in the management of adverse reactions, which should be guided by established recommendations. Healthcare providers hope to reduce allergic rhinitis's adverse effects on asthma, enhance the patient's quality of life, and diminish any related difficulties by treating the condition successfully.

ACKNOWLEDGEMENT:

Authors wishes to thanks Sankaralingam Bhuvaneswari College of Pharmacy, Anaikuttam, for providing the Library facility to complete this review study.

REFERENCES:

1. Wicz MS, Fineman S, Skoner DP, Nicklas R, Lee R, BlessingMoore J, *et al*. Diagnosis and management of rhinitis: complete guidelines of the Joint Task Force on Practice Parameters in Allergy, Asthma and Immunology. Ann Allergy Asthma Immunol, 1998; 81: 478-518.
2. Bousquet J, Neukirch F, Bousquet PJ, *et al.* Severity and impairment of allergic rhinitis in patients consulting in primary care. J Allergy Clin Immunol, 2006; 117: 158-162.
3. Walker S, Khan-Wasti S, Fletcher M, Cullinan P, Harris J, Sheikh A. Seasonal allergic rhinitis is associated with a detrimental effect on examination performance in United Kingdom teenagers: case-control study. J Allergy Clin Immunol, 2007; 120: 381-387.
4. Bousquet J, Khaltaev N, Cruz AA, *et al*. Allergic Rhinitis and its Impact on Asthma (ARIA) 2008. Allergy, 2008; 63 (suppl 86): 8-160.
5. Jacobsen L, Niggemann B, Dreborg S, *et al*. Specific immunotherapy has long-term preventive effect of seasonal and perennial asthma: 10-year follow up on the PAT study. Allergy, 2007; 62: 943-948.
6. Bostock J. Of the catarrhus æstivus, or summer catarrh. Med Chir Trans, 1828; 14: 437-446.
7. Mygind N, Anggard A, Druce H. Definition, classification, and terminology. In: Mygind N, Weeke B, editors. Allergic and vasomotor rhinitis. Copenhagen: Muunksgaard; 1985. pp. 15.
8. Fireman P. Allergic rhinitis. In: Fireman P, Slavin R, editors. Atlas of allergies. Philadelphia: JB Lippincott Co; 1991. pp. 9.2-9.18.
9. Wright AL, Holberg CJ, Martinez FD, Halonen M, Morgan W, Taussig LM. Epidemiology of physician-diagnosed allergic rhinitis in childhood. Pediatrics, 1994; 94: 895-901
10. Newacheck PW, Stoddard JJ. Prevalence and impact of multiple childhood chronic illnesses. J Pediatr, 1994; 124: 4
11. Von Mutius E, Martinez FD. Natural history, development, and prevention of allergic disease in childhood. In: Adkinson NF Jr, Yunginger JW, Busse WW, Bochner B, Holgate ST, Simons FER, editors. Middleton's allergy: principles and practice. 4th ed. St Louis: Mosby; 2003. pp. 1169-1174.
12. Scadding GK, Durham SR, Mirakian R, *et al*. BSACI guidelines for the management of allergic and non-allergic rhinitis. Clin Exp Allergy, 2008; 38: 19-42.
13. Von Mutius E. 99th Dahlem conference on infection, inflammation and chronic inflammatory disorders: farm lifestyles and the hygiene hypothesis. Clin Exp Immunol, 2010; 160: 130-135.
14. lks M. Allergy plants that cause sneezing and wheezing. Tampa: Worldwide Publications; 1986.
15. Platts-Mills TA, Hayden ML, Chapman MD, Wilkins SR. Seasonal variation in dust mite and grass-pollen allergens in dust from the houses of patients with asthma. J Allergy Clin Immunol, 1987; 79: 781-791.
16. Bradding P, Feather IH, Wilson S, Bardin PG, Heusser CH, Holgate ST, *et al.* Immunolocalization of cytokines in the nasal mucosa of normal and

perennial rhinitic subjects: the mast cell as a source of IL-4, IL-5, and IL-6 in human allergic mucosal inflammation. J Immunol, 1993; 151: 3853-3865.
17. Aberg N. Asthma and allergic rhinitis in Swedish conscripts. Clin Exp Allergy, 1989; 19: 59-63.
18. Tang RB, Tsai LC, Hwang HM, Hwang B, Wu KG, Hung MW. The prevalence of allergic disease and IgE antibodies to house dust mite in schoolchildren in Taiwan. Clin Exp Allergy. 1990; 20: 33-38.
19. Skoner D. Allergy and immunology. In: Zitelli B, Davis H, editors. Atlas of pediatric physical diagnosis. 3rd ed. New York: Mosby-Wolfe; 1997. pp. 75-110.
20. Mygind N, Anggard A, Druce H. Definition, classification, and terminology. In: Mygind N, Weeke B, editors. Allergic and vasomotor rhinitis. Copenhagen: Muunksgaard; 1985. pp. 15.
21. Evans R. Epidemiology and natural history of asthma, allergic rhinitis, and atopic dermatitis (eczema). In: Middleton E, Reed C, Ellis E, editors. Allergy: principles and practice. 4th ed. St Louis: Mosby; 1993. pp. 1109-1136.
22. Meltzer EO. Quality of life in adults and children with allergic rhinitis. J Allergy Clin Immunol, 2001; 8: S45-S53.
23. Hamilton RG, Franklin Adkinson N Jr. *In vitro* assays for the diagnosis of IgE-mediated disorders. J Allergy Clin Immunol, 2004; 114: 213-225.
24. Bousquet PJ, Chinn S, Janson C, Kogevinas M, Burney P, Jarvis D. Geographical variation in the prevalence of positive skin tests to environmental aeroallergens in the European Community Respiratory Health Survey I. Allergy, 2007; 62: 301-309.
25. Moscato G, Siracusa A. Rhinitis guidelines and implications for occupational rhinitis. Curr Opin Allergy Clin Immunol, 2009; 9: 110-115.
26. Scadding GK. Non-allergic rhinitis: diagnosis and management. Curr Opin Allergy Clin Immunol, 2001; 1: 15-20.
27. Malone DC, Lawson KA, Smith DH, *et al.* A cost of illness study of allergic rhinitis in the United States. J Allergy Clin Immunol, 1997; 99: 22-27.
28. Storms WW. Rethinking our approach to allergic rhinitis management. Ann Allergy Asthma Immunol, 2002; 88(suppl 1): 30-35.
29. Rice KD, Tanaka RD, Katz BA, Numerof RP, Moore WR. Inhibitors of tryptase for the treatment of mast cell-mediated diseases. Curr Pharm Des, 1998; 4:381-396.
30. Albert DH, Malo PE, Tapang P, *et al.* The role of plateletactivating factor (PAF) and the efficacy of ABT-491, a highly potent and selective PAF antagonist, in experimental allergic rhinitis. J Pharmacol Exp Ther, 1998; 284: 83-88.
31. Maurer M, Zuberbier T. Undertreatment of rhinitis symptoms in Europe: findings from a cross-sectional questionnaire survey. Allergy, 2007; 62: 1057-1063.
32. Bousquet J, Bodez T, Gehano P, *et al.* Implementation of guidelines for allergic rhinitis in specialist practices: a randomized pragmatic controlled trial. Int Arch Allergy Immunol, 2009; 150: 75-82.
33. Scadding GK, Durham SR, Mirakian R, *et al.* BSACI guidelines for the management of allergic and non-allergic rhinitis. Clin Exp Allergy, 2008; 38: 19-42.
34. Weiner JM, Abramson MJ, Puy RM. Intranasal corticosteroids versus oral H1 receptor antagonists in allergic rhinitis: systematic review of randomised controlled trials. BMJ, 1998; 317: 1624-1629.
35. Wilson AM, Orr LC, Sims EJ, Lipworth BJ. Effects of monotherapy with intra-nasal corticosteroid or combined oral histamine and leukotriene receptor antagonists in seasonal allergic rhinitis. Clin Exp Allergy, 2001; 31: 61-68.
36. Scadding G, Erkan AN, Chau H, Maskell S. Audit of nasal steroid use and effectiveness in a rhinitis clinic. Expert Rev Pharmacoecon Outcomes Res, 2010; 10: 87-90.
37. Boulet LP, Morin D, Milot J, Turcotte H. Bronchial responsiveness increases after seasonal antigen exposure in non-asthmatic subjects with pollen-induced rhinitis. Ann Allergy, 1989; 63: 114-119.
38. Löwhagen O, Rak S. Modifi cation of bronchial hyperreactivity after treatment with sodium cromoglycate during pollen season. J Allergy ClinImmunol, 1985; 75: 460-467.
39. Bousquet J, van Cauwenberge P, Ait Khaled N, *et al.* Pharmacologic and anti-IgE treatment of allergic rhinitis ARIA update (in collaboration with GA2LEN). Allergy, 2006; 61: 1086-1096.
40. Church MK, Maurer M, Simons FE, *et al.* Risk of first-generation H(1)-antihistamines: a GA(2)LEN position paper. Allergy, 2010; 65: 459-466.
41. Nasser SM, Ewan PW. Lesson of the week: depot corticosteroid treatment for hay fever causing

avascular necrosis of both hips. BMJ, 2001; 322: 1589-1591.
42. Dykewicz MS, Fineman S, Skoner DP, *et al.* Diagnosis and management of rhinitis: complete guidelines of the Joint Task Force on Practice Parameters in Allergy, Asthma, and Immunology. Ann Allergy Asthma Immunol, 1998; 81(Pt 2): 478-518.
43. Calderon MA. Meta-analyses of specific immunotherapy trials. Drugs Today (Barc), 2008; 44 (suppl B): 31-34.
44. Larenas-Linnemann D. Sublingual immunotherapy in children: complete and updated review supporting evidence of effect. Curr Opin Allergy Clin Immunol, 2009; 9: 168-176.
45. James LK, Durham SR. Update on mechanisms of allergen injection immunotherapy. Clin Exp Allergy, 2008; 38: 1074-1088.
46. Durham SR, Emminger W, Kapp A, *et al.* Long-term clinical efficacy in grass pollen-induced rhinoconjunctivitis after treatment with SQ-standardized grass allergy immunotherapy tablet. J Allergy Clin Immunol, 2010; 125: 131-138.
47. Scadding GW, Shamji MH, Jacobson MR, *et al.* Sublingual grass pollen immunotherapy is associated with increases in sublingual Foxp3-expressing cells and elevated allergen-specific immunoglobulin G4, immunoglobulin A and serum inhibitory activity for immunoglobulin E-facilitated allergen binding to B cells. Clin Exp Allergy, 2010; 40: 598-606.
48. Kuo CH, Wang WL, Chu YT, Lee MS, Hung CH. Sublingual immunotherapy in children: an updated review. Pediatr Neonatol, 2009; 50: 44-49.
49. Fokkens WJ, Lund VJ, Mullol J, Hopkins C, Reitsma S, Bachert C, *et al*. European Position Paper on Rhinosinusitis and Nasal Polyps group. Rhinol, 2020; 58(Suppl S29): 1-464.
50. Canonica GW, Blasi F, Carpagnano GE, Guida G, Heffler E, Paggiaro P, *et al*. Severe Asthma Network Italy definition of clinical remission in severe asthma: a Delphi consensus. J Allergy Clin Immunol Pract, 2023; 11(12): 3629-3637.
51. Ghosh A, Robbins K, Kelly J. The Cochrane Library: a resource for current reviews of clinical evidence. Minn Med, 2000; 83(7): 43-45.
52. Durham SR, Shamji MH. Allergen immunotherapy: past, present and future. Nat Rev Immunol, 2023; 23(5): 317-328.
53. Storms WW. Rethinking our approach to allergic rhinitis management. Ann Allergy Asthma Immunol, 2002; 88(suppl 1): 30-35.
54. Juniper EF, Guyatt GH, Ferrie PJ, Griffith LE. First-line treatment of seasonal (ragweed) rhinoconjunctivitis. CMAJ, 1997; 156: 1123-1131.

Conflict of Interest: None
Source of Funding: Nil
Paper Citation: Vigneswaran R*, Natarajan P, Thiruppathi M, Ganesh H, Kodishwaran R. A review on Allergic rhinitis with little insights of Diagnostic procedure and management. J Pharm Adv Res, 2024; 7(3): 2127-2134.